Type V Gastric Ulcer: An Unusual Etiology of Gastrosplenic Fistula Associated with Upper Gastrointestinal Bleeding

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Introduction

Gastrosplenic fistula (GSF) is a rare entity characterized by an abnormal communication between the gastric cavity and the spleen. Reported etiologies of GSF are splenic or gastric malignancies, splenic abscess, crohn's disease, peptic ulcers disease, sickle cell disease, sleeve gastrectomy and abdominal trauma[1,2]. Diffuse large B cell lymphoma (DLBCL) is the most common malignancy resulting in GSF[1,2]. Predominant symptoms at clinical presentation are abdominal pain, hematemesis, melena, and constitutional symptoms such as generalized weakness, fever and weight loss. Imaging findings of air bubbles and leakage of oral contrast from gastric cavity into the spleen is diagnostic of GSF[3]. Prognosis and short-term survival in patients with GSF are good (up to 82%) in patients presented without gastrointestinal bleeding (GIB)[4]. A small proportion of patients with GSF may present with life-threatening GIB. We present a case of GSF secondary to type V gastric ulcer associated with upper GIB.

Case History

A 41-year-old man with a history of oculodentodigital dysplasia, spastic paraplegia and sacral decubitus ulcers with diverting sigmoid colostomy, chronic back pain on baclofen pump presented with malaise and melena through colostomy. He admitted intermittent use of NSAIDs for chronic back pain. On arrival to ER, he was tachycardic (113/min), with a BP of 126/82 mmHg and pale appearing. His Hgb was 4.9 g/dL (baseline Hgb 12.1 g/dL). CT angiogram of the abdomen/pelvis was negative for active contrast extravasation, however, revealed direct contiguity between the gastric fundus and spleen indicating GSF (figure 1). The patient was resuscitated with IV fluid and required multiple blood transfusions. Splenic artery (SA) angiogram showed hyperemia along the posterior wall of the stomach corresponding to CT findings of GSF (figure 2). Embolization of the main SA, right gastroepiploic, and left omental artery was performed (figure 3). An EGD showed a benign inflammatory mass in the gastric fundus with the invasion of splenic tissue into the gastric mucosa (figure 4). A gastric biopsy was negative for H. pylori and malignancy, however, revealed mild chronic inflammation and reactive gastropathy. Patient was managed with partial gastrectomy and splenectomy. Operative findings were consistent with a large type V gastric ulcer at the fundus with direct extension into the spleen. Pathology of operative specimen demonstrated transmural granulation tissue and acute inflammation with acute serositis and mucosal ulceration without evidence of ischemia. He was discharged to rehab after a prolonged hospital course.

Discussion

Gastrosplenic fistula due to type V gastric ulcer complicated by massive GIB is rare. There are five types of benign gastric ulcers including true gastric ulcers at incisura angularis (type I), combined gastric and duodenal ulcers (type II), Pyloric and pre-pyloric ulcers (type III), ulcers high along lesser curvature within 1-2cm of gastroesophageal junction (type IV) and gastric ulcers anywhere secondary to medications (type V)[5]. The incidence of type V ulcers is <5%. The proposed pathogenic mechanism of GSF are progressive infiltrative, erosive, and penetrating lesions of spleen and/or adjacent gastric wall. A subset of patients with GSF, present with GIB due to necrosis of underlying blood vessel in splenic parenchyma, gastric wall and gastrosplenic ligament that contains short gastric, and left gastroepiploic vessels[6].

In our case the patient had significant drop in hemoglobin from his baseline and evidence of GSF on cross-sectional radiologic images that was the source of GIB. Abdominal CT was negative for splenic abscess, ischemia, lymphadenopathy or mass concerning for malignancy. Although patient was hemodynamically stable on admission and there was no active bleeding on CT scan, an embolization of the main splenic artery, right gastroepiploic, and left omental artery was performed by interventional radiology prior to EGD to prevent procedural related risks of iatrogenic bleeding as biopsy of gastric ulcer was essential to rule out malignancy and other etiologies of GSF[7]. Gastric biopsies were negative for H-pylori, malignancy, however demonstrated non-specific findings of mild chronic inflammation and reactive gastropathy that may be seen

in patients with GSF and its sequalae. Operative findings and pathology of operative specimen demonstrated transmural granulation tissue and acute inflammation with acute serositis and mucosal ulceration consistent with type V gastric ulcer. The risk factor of type V gastric ulcer in our patient was chronic NSAID's use intermittently for back pain which may results in fistulation due to chronic inflammation involving transmural gastric wall ulceration.

Prior to definitive surgical management of GSF, an EGD evaluation of other causes of upper GIB is crucial to establish etiology by obtaining tissue diagnosis. Surgical resection of GSF is curative treatment. The choice of surgical resection depends on patient's hemodynamic stability, extension of underlying disease and surgeons' preferences. Laparoscopic partial gastrectomy with or without splenectomy is common method of surgical resection with favorable outcomes[1,2,8]. Non-surgical management of GSF with chemotherapy has also been reported effective in several cases of DLBCL[8-10].

Conclusions

Type V gastric ulcer is a rare etiology of GSF that may be overlooked cause of GIB. Early embolization of splenic vessels prior to EGD and surgical resection is crucial to minimize procedure related complication and post-procedural outcomes. A high index of clinical suspicion is required for early identification and management of GSF as massive hemorrhage results in high rate of mortality.

Authorship list:

Muhammad Nadeem Yousaf: Made the most significant contribution in manuscript preparation including drafting, writing, revisions and final approval.

Riyasha Dahal: Contributed to literature review, reviewing the article, revision and final approval.

Subeena Phull: Made the most significant contribution in writing and clinical case data gathering.

Aryal Vinayak, Karun Neupane, Hamza Ertugrul contributed in literature review and data gathering.

Ebubekir Daglilar: contributed in overall supervision of and review of manuscript.

All authors read and approved the final manuscript

Figures legends:

Figure 1: CT angiogram shows wedge shaped defect between gastric fundus and spleen indicating gastrosplenic fistula (arrow).

Figure 2:

Catheter angiography of the splenic artery shows extravasation of blood along the posterior wall of the stomach corresponding to the site of gastric ulceration on CT scan.

Figure 3:

Splenic artery angiogram after Gelfoam slurry, coil, and Amplatzer embolization. There is no contrast extravasation after embolization.

Figure 4:

EGD shows a large benign gastric ulcerated mass in the gastric fundus, consistent with splenic tissue invading through gastric wall ulcer.

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